Amendments to the Specification:

Please replace paragraph 66 with the following amended paragraph:

[66] The terms "GPCR" and "TGR342", "TGR60", "TGR346", and "TGR399" ~~"TGR-342, -60, -346, and 399" therefore~~ refer to polymorphic variants, alleles, mutants, and interspecies homologs and GPCR domains thereof that: (1) have about 70% amino acid sequence identity, preferably about 75, 80, 85, 90 or 95% or higher amino acid sequence identity, to SEQ ID NO:2; SEQ ID NO:4, SEQ ID NO:6, SEQ ID NO:8, SEQ ID NO:10, SEQ ID NO:12, SEQ ID NO:16, or SEQ ID NO:18 over a window of about 25 amino acids, preferably 50-100 amino acids; (2) bind to antibodies raised against an immunogen comprising an amino acid sequence of SEQ ID NO:2; SEQ ID NO:4, SEQ ID NO:6, SEQ ID NO:8, SEQ ID NO:10, SEQ ID NO:12, SEQ ID NO:16, or SEQ ID NO:18 and conservatively modified variants thereof; (3) specifically hybridize (with a size of at least about 100, preferably at least about 500 or 1000 nucleotides) under stringent hybridization conditions to a sequence SEQ ID NO:1; SEQ ID NO:3, SEQ ID NO:5, SEQ ID NO:7, SEQ ID NO:9, SEQ ID NO:11, SEQ ID NO:15, or SEQ ID NO:17, and conservatively modified variants thereof; or (4) have a nucleic acid sequence that has greater than about 95%, preferably greater than about 96%, 97%, 98%, 99%, or higher nucleotide sequence identity, preferably over a region of at least about 50, 100, 200, 500, 1000, or more nucleotides, to SEQ ID NO:1; SEQ ID NO:3, SEQ ID NO:5, SEQ ID NO:7, SEQ ID NO:9, SEQ ID NO:11, SEQ ID NO:15, or SEQ ID NO:17; (5) are amplified by primers that specifically hybridize under stringent conditions to SEQ ID NO: SEQ ID NO:1; SEQ ID NO:3, SEQ ID NO:5, SEQ ID NO:7, SEQ ID NO:9, SEQ ID NO:11, SEQ ID NO:15, or SEQ ID NO:17. This term also refers to a domain of a GPCR, as described above, or a fusion protein comprising a domain of a GPCR linked to a heterologous protein. A TGR-342, -60, -346, or -339 protein or domain typically comprises 10, 15, often 20, 25, or 30 or more contiguous amino acids of SEQ ID NO:2, 4, 6, 8, 10, or 12. A TGR-342, -60, -346, or TGR-339 nucleic acid typically comprises at least 15, often 20, 25, 30, or 50 or more contiguous nucleotides of a sequence of SEQ ID NOs: 1, 3, 5, 7, or 9. GPCR polynucleotide or polypeptide sequence of the invention is typically from

a mammal including, but not limited to, human, rat, mouse, hamster, cow, pig, horse, sheep, or any mammal. A "TGR-342, -60, -346, and -339 polynucleotide" and a "TGR-342, -60, -346, and –339 polypeptide," are both either naturally occurring or recombinant.

Please replace paragraph 7 with the following amended paragraph:

[07] GPCRs are also involved in retinal function and additionally may play an important role in the pathology of retinal disease. *Retinitis pigmentosa* is a retinal degeneration characterized by the following manifestations: night blindness, progressive loss of peripheral vision, eventually leading to total blindness; ophthalmoscopic changes consist in dark mosaic-like retinal pigmentation, attenuation of the retinal vessels, waxy pallor of the optic disc, and in the advanced forms, macular degeneration. In some cases there can be a lack of pigmentation. *Retinitis pigmentosa* can be associated and degenerative opacity of the vitreous body, and cataract. A number of more complex syndromes are often associated to this disease, such as Usher's syndrome, responsible for deafness; Laurence-Moon syndrome, characterized by hypogonadism, mental retardation and obesity; Refsum's syndrome which can lead to mental retardation and dwarfism. Family history is prominent in *retinitis pigmentosa*; the pattern of inheritance may be autosomal recessive, autosomal dominant, or X-linked; the autosomal recessive form is the most common and can occur sporadically. Disease incidence varies from 1/2000 to 1/7000 according to the type of investigation and geographic location. Although r*etinitis pigmentosa* was first described a century ago; its pathogenesis is, nevertheless, still unknown. (*see*, ~~Molecular Genetic Investigations of Eye Disease, http://ucl.ac.uk/ioo/research/bhattacharya.htm, and~~ den Hollander, *Nature Genetics*, 23:217-221 (October 1999), the teachings ~~of both~~ of which are incorporated herein by reference).